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13. ABSTRACT *(Maximum 200 words)*

This study determined if hypohydration alters heart rate variability (HRV) and its autonomic control before and after exercise-heat stress. Five heat-acclimated subjects performed exercise-heat trials (40oC, 20% relative humidity), once when euhydrated and once when hypohydrated (3.9 ± 0.7% body weight loss) in a counterbalanced order. During each trial, subjects sat quietly for 45 min before completing 90-min of cycle ergometer exercise, which was followed by 45 min of inactive recovery. Cardiac cycle R-R interval data were collected for 45 min pre and post exercise exposures and analyzed by Fast Fourier Power Spectral analysis to determine the high frequency (HF), low frequency (LF), very low frequency (VLF) and Total Power (TP) components of HR variability. Before and after exercise, TP, LF, VLF, LF:HF ratio were lower ($P<0.05$) while HF was higher when hypohydrated. Our data indicate that: 1) heart rate variability is decreased by both hypohydration and exercise-heat stress; and 2) hypohydration increases the parasympathetic control of heart rate variability; and 3) this parasympathetic influence is augmented by exercise-heat stress. These data suggest that an increased parasympathetic influence on heart rate variability when hypohydrated may act to reduce the risk of cardiac dysrhythmia.

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The influence of hydration status on heart rate variability after exercise heat stress

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Abstract

While exercise heat stress and hydration status are known to independently influence heart rate variability (HRV), the combined effect of these physiological stressors is unknown. Thus, heat-acclimated subjects ($n = 5$) performed exercise heat trials (40 °C, 20% relative humidity) in the euhydrated and hypohydrated state (3.9 ± 0.7% body weight loss). During each trial, cardiac cycle R–R interval data were collected for 45 min at rest (pre-) and after (post-) completing 90 min of cycle ergometer exercise. Pre- and post-exercise RRI data were analyzed by Fast Fourier Power Spectral analysis to determine the high-frequency (HF), low-frequency (LF), very low-frequency (VLF), and total power (TP) components of HRV. Overall HRV was decreased by both hypohydration and exercise heat stress. Hypohydration reduced TP, LF, VLF, and LF:HF ratio ($P < 0.05$) while HF was significantly higher. The change in both LF and HF power (pre- vs. post-exercise) were blunted during hypohydration compared to euhydration. These data suggest that dehydration alone positively influences the parasympathetic (HF) control of HRV, but the reduction in overall HRV and the blunted oscillations in LF and HF power following exercise heat stress support an overall deleterious effect of dehydration on autonomic cardiac stability.
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Keywords: Heart rate variability; Autonomic nervous system; Exercise; Recovery

1. Introduction

Exercise-induced tachycardia results primarily from parasympathetic withdrawal at low exercise intensities and from both parasympathetic withdrawal and sympathetic stimulation at moderate (Robinson et al., 1966) and high exercise intensities (Nordenfelt, 1971). However, during recovery from exercise, the mechanisms which lower heart rate (HR) (i.e. cardio-deceleration) are less well understood. Inactive recovery from exercise is associated with the cessation of the primary feed-forward exercise stimulus from the brain (central command), which is responsible for the initial rapid deceleration in HR (Carter et al., 1999). Other factors influencing autonomic control of cardiac deceleration may include changes in afferent stimuli from muscle metabo- and mechanoreceptors (Wilson et al., 2004), baroreceptors, and thermoreceptors (Fisher et al., 1999). Nevertheless, the robust return of parasympathetic

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activity is considered to be the main mechanism underlying cardio-deceleration after exercise (Carter et al., 1999; Nishime et al., 2000).

The degree of autonomic control of HR during recovery from exercise may be altered by hydration status and body temperature. Body water deficits (hypohydration) have been associated with increased cardiovascular strain (Sawka and Coyle, 1999) and under very severe conditions, cardiac dysrhythmia and arrest may occur (MMWR, 1998). Hypohydration-mediated cardiovascular strain results primarily from reduced plasma volume and redistribution of blood to warm skin due to hyperthermia (Sawka and Coyle, 1999). Also, passive hyperthermia alone (which is augmented by exercise and hypohydration) is reported to decrease cardiac vagal modulation of HR (Crandall et al., 2000). Despite the well-characterized effects of hypohydration on cardiovascular and heat strain, its influence on autonomic control of HR has not been reported. Hypohydrated subjects can have altered muscle metabolism, reduced baroreceptor responsiveness (Charkoudian et al., 2003), difficulty sustaining arterial blood pressure (Gonzalez-Alonso et al., 1997), elevated circulating catecholamines and greater hyperthermia (Sawka et al., 1985) both during and after exercise heat stress. Each of these factors may presumably influence the autonomic control of HR.

Power spectral analysis of heart rate variability (HRV) has proved to be a reliable, non-invasive method for quantitative assessment of cardiac sympathetic and parasympathetic nervous system activity in humans (Matsumoto et al., 1999; Perini and Veicsteinas, 2003b). In general, the power spectra can be divided into low-frequency (LF) and high-frequency (HF) components (Perini and Veicsteinas, 2003a). While some debate remains regarding the interpretation of these frequency components, it is generally believed that the LF power is modulated by both sympathetic and parasympathetic outflow (Akselrod et al., 1981; Pomeranz et al., 1985), while the HF power reflects primarily parasympathetic influence (Chiu et al., 2003). The physiological basis for such an assessment is that the cardiac response to parasympathetic activity is more rapid than sympathetic activity (Perini and Veicsteinas, 2003a). Thus, quantitative information concerning autonomic control of HR may be obtained from HRV spectral analysis in various conditions (Saul et al., 1990; Perini and Veicsteinas, 2003b). Previous studies have shown that after exercise, HF HRV is reduced (Hautala et al., 2001) and that additional perturbations of hypoxia accentuate the sympathetic dominance during rest and exercise (Buchheit et al., 2004). However, the combined influence of hypohydration and exercise heat stress on HRV has not been reported.

Thus, the current study sought to determine the effect of hydration status on HRV before and after exercise-induced heat stress. We hypothesized that (1) hypohydration alone would increase sympathetic and decrease parasympathetic cardiac activity, resulting in a decrease in resting HRV, and that (2) hypohydration in combination with exercise-induced heat stress would further decrease overall HRV, leading to autonomic instability.

2. Methods

2.1. Subjects

Five healthy male volunteers (age 23 ± 4 (SD) yr, body mass 70.7 ± 7.0 kg, and body fat $17.1 \pm 4.6\%$) participated in this study and completed both trials. Subjects were physically active and moderately fit (peak oxygen uptake ($\dot{V}O_{2peak}$) of 48 ± 4 ml/kg/min). The appropriate Institutional Review Boards approved this study after all subjects gave their voluntary written informed consent to participate.

2.1.1. Preliminary procedures

Aerobic fitness. $\dot{V}O_{2peak}$ was measured on an electronically braked cycle ergometer (Lode Excalibur Sport, Groningen, Netherlands) interfaced with a computer-based metabolic system (TrueMax, ParvoMedics, Sandy, Utah). Exercise began at an initial power output of 100 W for 5 min, followed by increments of 30 W every 2.5 min until volitional exhaustion, which was defined as an inability to maintain the designated pedal cadence (60 ± 5 rpm).

Heat acclimation. Subjects were heat acclimated by walking on a treadmill (4% grade at 1.56 m/s) for two 50 min exercise bouts separated by a 10 min rest for 9 days. The environment was identical to that chosen for experiments (40 °C, 20% RH, wind speed 1 m/s). Semi-nude body mass (shorts only) was measured after voiding and before breakfast each morning (9 days) to establish a mean individual baseline body mass representative of a euhydrated state.

2.2. Experimental testing

2.2.1. Design

Subjects completed two experimental trials in counterbalanced order. Each trial included one preparation day (day 1) and one test day (day 2). The afternoon of day 1, 2–3 h of intermittent treadmill walking and running was performed in the heat (40 °C, 20% RH) and drinking was restricted to produce a 4% loss of body mass (hypohydration trial) or water was administered ad libitum to maintain body mass (euhydration trial). Subjects then showered and ate a standardized meal before spending a supervised night in a comfortable environment. Overnight non-food fluid intake was

restricted to 200 ml during the hypohydration trials. These hypohydration procedures are consistent to those previously employed in our laboratory (Sawka et al., 1992). On the morning of day 2, subjects were instrumented and then sat in the heat for 45 min before completing a 90 min of cycle ergometer exercise at ~60% VO_{2peak}. After completion of the 90 min exercise bout, subjects recovered (seated) for 45 min in the same environment. All experiments were conducted at the same time of the day to control for circadian patterns in HR and thermoregulation (Stephenson et al., 1984).

2.2.2. Procedures

Body mass was measured semi-nude with an electronic precision balance scale before and after each experimental trial. The mean baseline body mass was used to calculate the precise fluid deficit. Oxygen uptake was determined by open-circuit spirometry at 25 min intervals during exercise. The average exercise intensity (% VO_{2peak}) elicited in trial 1 was replicated in trial 2. Rectal temperature (T_{re}) was continuously obtained from a thermistor inserted 10 cm beyond the anal sphincter. A 6% commercial carbohydrate–electrolyte drink was given at 15 min intervals throughout exercise to approximate estimated sweat losses (1 l/h) and maintain plasma glucose. RPE was measured using the Borg scale.

2.3. Blood analysis

Venous blood samples were collected into ice-chilled tubes before and after exercise testing through an indwelling plastic catheter in a superficial arm vein. Resting, pre-exercise blood samples (10 ml) were drawn in the fasting state (~10 h) after subjects had been instrumented. An additional 10 ml was drawn immediately post-exercise while still seated on the cycle ergometer with the same arm position. Patency was maintained using non-heparinized saline (0.9% w/v). Prior to the collection of each sample, ~3 ml of fluid was withdrawn and discarded to clear the catheter dead space. Plasma lactate was measured using immobilized enzyme electrochemical biosensors (YSI Inc., Yellow Springs, OH). Plasma osmolality was measured by freezing point depression.

2.4. Data analysis

2.4.1. Spectral analysis of HRV

The R–R series (interval tachogram) was obtained from the polysomnographic ECG recording with a sampling frequency of 512 Hz (Embla, Flaga, Inc) and computed with an algorithm of R-wave recognition (derivative + threshold). Each tachogram was then divided into segments according to time (10 min pre-exercise and 10 min post-exercise intervals), excluding the first 15 min of recovery. Each segment was then searched for artifacts and for missing ectopic beats that could affect spectral estimation. Segments belonging to a given period lasting >256 consecutive beats were considered eligible for spectral analysis.

A Fast Fourier Transformation (FFT) Model (Nevrokard, Medistar, Inc. Solevenia) was used for the frequency domain variables of HRV. We computed total spectral power in the very low frequency (VLF, ≤0.04 Hz), low frequency (LF, 0.04–0.15 Hz), and high frequency (HF, 0.15–0.40 Hz), expressed in terms of (1) absolute units ($m\,s^2$), (2) percentage power (i.e., percent of the total spectral power), (3) normalized power (i.e., percentage of the total spectral power minus the VLF component), and (4) LF:HF ratio (i.e., LF/HF), see Table 1. As recommended by the Task Force of the European Society of Cardiology (1996) guidelines, absolute units of LF and HF were calculated for the purposes of comparison; however, absolute units were not used to evaluate the autonomic nervous system balance because the increased HR during sympathetic activation is usually accompanied by a reduction in total power (TP) (Task Force of the European Society of Cardiology, 1996). The normalization procedure is particularly helpful in allowing comparisons between subjects or experimental conditions characterized by large differences in TP or DC noise (Task Force of the European Society of Cardiology, 1996). HRV analyses were conducted on the final 10 min of the 45 min pre-exercise and the final 30 min of the 45 min post-exercise period. Only the final 30 min of the post-exercise period was assessed in order to allow breathing frequency to return to pre-exercise homeostasis.

Subjects breathing frequency was similar both before (13±1 breaths/min) and after (15±2 breaths/min) exercise, and was not altered by hydration status. Previous studies have demonstrated that HF HRV is similar with spontaneous and metronomic breathing (Patwardhan et al., 1995) and that small changes in breathing patterns do not alter spectral and transfer analyses of HR with passive heat exposure (Crandall et al., 2000).

2.4.2. Statistics

All data analyses were performed using Sigma Stat 2.2 (SPSS, Inc) statistical software and are presented as means±SD. Following tests for normality of distribution and equality of variances, treatment effects were analyzed using a two-way ANOVA (trial x time) for repeated measurements. When appropriate, Tukey's HSD procedure was used to identify differences among means following significant main and/or interaction effects. Effect sizes estimates were derived from power spectral data using conventional alpha and beta values. The Study Sample size was adequate to detect

Table 1
Frequency domain indexes (normalized values) before and after exercise heat stress

	Pre-exercise	25 min post-exercise	35 min post-exercise	45 min post-exercise
LF, n.u.				
Euhydration	53.4±24.5	79.9±26.5	77.7±25.4	68.3±21.5
Hypohydration	39.2±19.8	47.3±19.8	50.1±19.8	27.4±14.7
P value	<0.05	<0.05	<0.05	<0.05
HF, n.u.				
Euhydration	42.1±25.4	9.5±4.9	16.1±10.6	28.0±18.9
Hypohydration	55.6±16.5	48.5±24.7	46.4±15.4	64.7±24.5
P value	<0.05	<0.01	<0.05	<0.05
LF:HF				
Euhydration	1.4±0.6	8.40±2.5	4.8±1.5	2.5±0.8
Hypohydration	0.7±0.2	0.9±0.3	1.1±0.9	0.4±0.5
P value	0.05	0.01	0.01	0.05
VLF, n.u.				
Euhydration	27.9±16.5	36.9±14.9	44.5±21.4	25.7±19.2
Hypohydration	19.6±5.6	48.9±25.6	29.0±20.9	63.1±36.5
P value	<0.05	NS	<0.05	<0.05
TP (ms^2)				
Euhydration	3178±265	2714±394	2814±212	2913±231
Hypohydration	2278±389	1889±436	1963±298	2016±269
P value	<0.01	<0.01	<0.05	<0.05

LF, low frequency; HF, high frequency; VLF, very low frequency; TP; total power.

meaningful physiological differences in HRV. (Significance accepted at $P<0.05$.)

3. Results

3.1. Hydration and blood

Plasma osmolality was lower ($P<0.05$) prior to the start of the euhydration trial (284±2 mosmol/kg) compared to hypohydration (300±2 mosmol/kg) trial. The level of hypohydration achieved prior to the start of the hypohydration trial was 3.9±0.7% body mass loss. During exercise heat stress, sweat losses exceeded standardized fluid intakes so that total body mass losses after 90 min exercise were 1.6±0.3% (range = 1.3–2.1) for euhydration and 5.1±0.7% (range = 3.8–5.8) for hypohydration trials, respectively. There was no difference ($P>0.05$) between trials for post-exercise blood lactate levels (EUH = 3.9±3.5 vs. HYPO = 3.8± 2.1 mmol/l).

3.2. Exercise responses

The relative exercise intensity (59% VO_{2peak}) was similar ($P>0.05$) for the euhydration and hypohydration trials. Likewise, the RPE at the end of exercise heat stress was similar ($P>0.05$; 18±2 vs. 18±2). However, the total amount of work completed during the exercise bout was 11% greater ($P<0.05$) during the euhydration trial (501±79 kJ, range = 413–627 kJ) than hypohydration trial (449±70 kJ, range = 412–550 kJ).

Fig. 1 represents HR and rectal temperature responses during exercise and recovery from exercise over time. At rest, HR and rectal temperature values were higher ($P<0.05$) during hypohydration compared to the euhydration trial. During exercise, rectal temperature was higher ($P<0.05$) when hypohydrated, while HR was not different between trials. Recovery HRs and rectal temperature values were higher ($P<0.01$) when hypohydrated.

3.3. HRV indices

Table 1 provides the frequency domain indices before and after exercise in the euhydrated and hypohydrated state.

3.3.1. Hydration and autonomic control

The exclusive impact of hydration level on autonomic control of HRV was assessed in the pre-exercise period. In this period, hypohydration produced significantly lower ($P<0.05$) HRV values for TP, LF, VLF, and

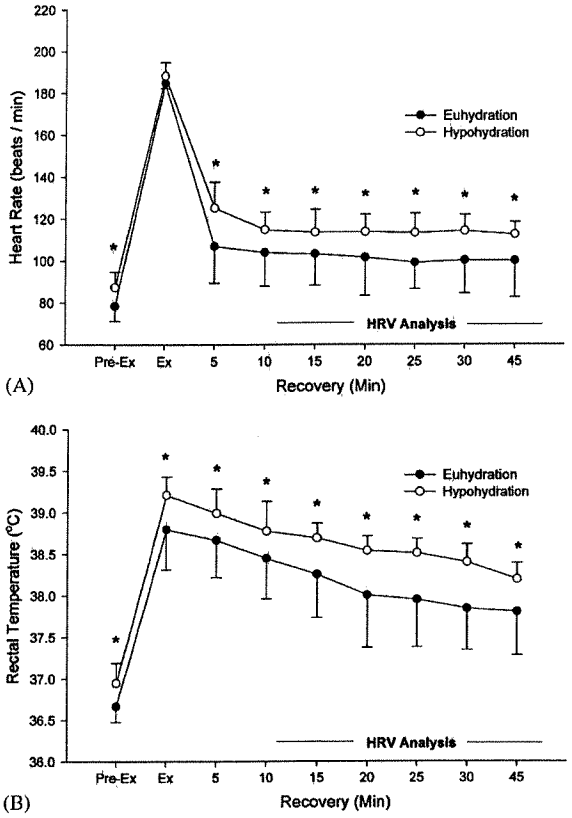

Fig. 1. HR and core temperature responses at rest, during and after exercise in the heat when euhydrated and hypohydrated. * indicates significant differences between the euhydration and hypohydration trials ($P<0.05$).

LF/HF ratio, while HF values were higher ($P<0.05$), Table 1.

3.3.2. Combined effect of hypohydration and exercise

The interaction of hydration status with exercise heat stress was accomplished by comparing pre- to 25 min post-exercise. Between these conditions, there was a greater reduction in HF values when euhydrated (−33 n.u. or −78%) than when hypohydrated (−7 n.u. or −13%). The LF power and TP were lower in the hypohydrated state compared to euhydration before exercise and during all points of exercise recovery (Table 1). As with HF values, changes in LF power (pre- vs. 25 min post-exercise) were significantly greater in the euhydrated state (+26 n.u. or +50%) compared to the dehydrated state (+8 n.u. or +20%).

4. Discussion

This study is the first to demonstrate that hypohydration significantly reduces HRV at rest and during recovery from exercise heat stress. Frequency domain analysis of low (LF) and high (HF) frequency power revealed a shift towards greater parasympathetic influence of the heart during hypohydration, which may serve to limit the cardiovascular stress induced by dehydration. However, fluctuations in LF and HF power during recovery from exercise were blunted in the hypohydrated state, suggesting that hydration status contributes importantly to restoration of autonomic balance following exercise heat stress. Together, these data suggest that dehydration alone positively influences parasympathetic control of HRV, but the reduction in overall HRV and the diminution of LF and HF changes following exercise results in an overall deleterious effect of dehydration on autonomic cardiac stability.

4.1. HRV and cardiac risk

In the current study, hypohydration and exercise heat stress both markedly reduce the TP of HRV. When subjects initiated the hypohydration exercise heat trial, they had been dehydrated ~15 h prior and had rested quietly in a temperate climate. Despite this extensive period of rest, they demonstrated decreased LF, LF:HF ratio, and TP, clearly demonstrating that hypohydration can have prolonged effects on reducing HRV. Subsequent exercise heat stress accentuated the reduction in HRV which was sustained for the 45 min recovery period (Table 1).

HRV is increasingly used clinically as a non-invasive indicator of cardiovascular health, and may serve as an independent predictor of sudden cardiac death (Molgaard et al., 1991). Others have demonstrated that reduced HRV values are associated with increased risk of cardiac events including dysrhythmia (1996) and post-infarction mortality (Wichterle et al., 2004). While many of these clinical studies fail to identify specific mechanisms which link HRV with adverse cardiac events, this well-established correlation provides the impetus for studies which seek to identify physiologic conditions which alter HRV and thus may influence overall cardiac health.

High-intensity exercise heat stress has also been associated with increased risk of subsequent cardiac events (Haskell, 1982). Previous studies have documented that exhaustive exercise will also reduce HRV (Arai et al., 1989; Hautala et al., 2001). The present study extends these findings by identifying hydration status as an important contributor to overall HRV at rest and following exercise heat stress. Thus, based on the known negative correlation between HRV and cardiac risk, our findings of a hypohydration-induced reduction in HRV at rest and following exercise indicates the potential for a hydration-associated increased risk of cardiac events.

4.2. Autonomic adaptations to heat and hypohydration

Using passive heating with very high skin temperatures but with lower core temperatures, Crandall and colleagues (Crandall et al., 2000) reported that HRV (including the HF range) was reduced, while others have reported that exercise and exercise with hypoxia will reduce both TP and HF HRV (Arai et al., 1989). Control data from the present study support these findings, as we observed that HF oscillations were decreased in the euhydrated state following exercise heat stress. In contrast, the resting HF HRV was increased in the hypohydration state, and remained high following exercise. Interestingly, the change in both LF and HF power from pre- to post-exercise were blunted in the hypohydrated state (Fig. 2), suggesting a reduction in autonomic regulatory capacity to the combined physiologic strain of dehydration and exercise heat stress. In conjunction with the overall diminution in HRV during hypohydration, this apparent lack of autonomic adaptation may indicate an inability to achieve autonomic cardiac stability in the hypohydrated, heat-stressed state following exercise.

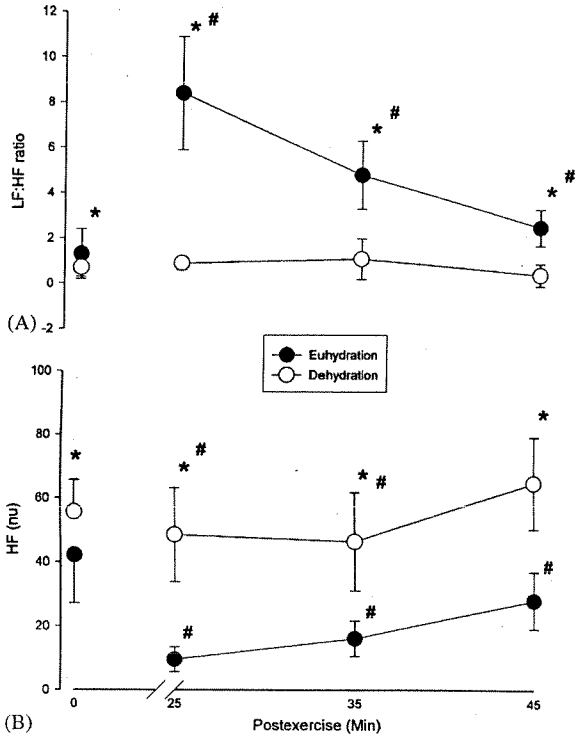

Fig. 2. LF:HF ratio and HF values, before and after 90 min exercise heat stress. * indicates significant differences between the euhydration and hypohydration trials; # indicates significant differences in time by trial from pre-exercise values. HF = high frequency and LF:HF ratio = low-frequency to high-frequency ratio.

Charkoudian et al. (2003) recently examined the influence of hypohydration on cardiovascular control after exercise heat stress. Subjects underwent a nitroprusside (exogenous nitric oxide donor) and phenylephrine (alpha-adrenergic agonist) challenge to evaluate baroreflex gain 90 min after completing exercise heat stress. When subjects were hypohydrated by 1.6% body weight loss, cardiac baroreceptor gain was diminished compared to when euhydrated. In addition, saline infusion did not ameliorate this reduction in baroreceptor gain. These data provide evidence that cardiac baroreflex changes contribute, not protect, persons from the orthostatic intolerance observed when hypohydrated. While this study did not measure HRV, these findings clearly emphasize the impact of hydration status on overall autonomic control, and lend support to our observation that hypohydration reduces the fluctuations in LF and HF power following heat stress exercise.

Changes in HRV reported herein may reflect compensatory cardiac parasympathetic outflow and decreased cardiac baroreflex sensitivity (Charkoudian et al., 2003), which may be a consequence of post-exercise hypohydration. Noteworthy work has been completed by Javorka et al. (Javorka et al., 2002) and others (Arai et al., 1989; Cole et al., 1999) addressing post-exercise HRV; however, those studies focused on the effects of exercise per se and did not examine the influence of hyperthermia or hypohydration on HRV during exercise recovery. They demonstrated that increased HF component of HRV contributes importantly to cardiodeceleration during the early phases of the post-exercise recovery. Data from the current study extend these findings to include the role of hydration status before and after exercise heat stress.

4.3. Proposed mechanisms of increased parasympathetic control

The increased HF oscillation in HRV with hypohydration alone strongly suggests an increased parasympathetic component (Saul, 1990; Perini, 2003). Although the mechanism responsible for this increase in the HF component of HRV is unknown, autonomic adaptations involving changes in central, cardioreflex, and/or end organ activity or responsiveness are likely candidates (Chiu et al., 2003). While the hypohydration level had been established for several hours, this autonomic adaptation persisted, as altered HRV parameters were sustained for more than 15 h after dehydration sessions and more than 45 min after exhaustive exercise heat stress. Thus, while we observed that hydration status and exercise provoke lasting changes in HF HRV, the current data do not address the exact mechanisms responsible for this intriguing autonomic adaptation.

Charkoudian et al. (Charkoudian et al., 2003) showed that even with the restoration of plasma volume with

saline infusion, autonomic control was not immediately restored in dehydrated subjects, further evidence of dehydration-induced changes in autonomic function. Hautala et al. (2001) reported that after a 75 km skiing race, HRV and HR were reduced for more than 7 h. They showed that the HF spectral component of HRV was lower in the first day after the race compared with pre-exercise values, suggesting that cardiac vagal outflow was attenuated. However, hydration status was not assessed in this study. We believe that inclusion of multiple perturbations in the present study provides novel information about cardiac autonomic regulation during recovery from exercise in dehydrated individuals.

4.4. Limitations

The present experiments were carefully controlled regarding hydration status and exercise heat exposure. Accordingly, the exercise heat exposure employed a constant load exercise bout so that exercise duration did not vary and both induced similar physical strain (similar RPE data). However, because relative exercise intensities were the same in both trials, 11% more work was performed in the euhydration trial due to the well documented effect of hypohydration on reducing work output (Cheuvront et al., 2003). In addition, the rate and depth of breathing were not controlled in the present study, parameters which might alter HF oscillations of HR. However, others have shown similar HF power of HR between metronomic and spontaneous breathing (Patwardhan et al., 1995), and in the current study spontaneous breathing rate did not differ between trials.

4.5. Summary

Findings from the current study indicate that: (1) overall heart rate variability (HRV) is decreased by both hypohydration and exercise heat stress; (2) hypohydration increases the parasympathetic control of HRV, indicated by an increase in high frequency (HF) HRV; and (3) this parasympathetic influence is further augmented by exercise heat stress. These data suggest that dehydration alone positively influences parasympathetic control of HRV, but the reduction in overall HRV and the blunted oscillations in LF and HF power following exercise suggest an overall deleterious effect of dehydration on autonomic cardiac stability.

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